Message

From: Caldwell, Jane [Caldwell.Jane@epa.gov]
Sent: 2/27/2015 9:21:25 PM
To: Fritz, Jason [Fritz.Jason@epa.gov]
Subject: FW: FA literature
Attachments: Kirsch-volders et al 2014 lymphocyte cytokinesis-block micronucleus assay (1).pdf; Yu et al 2014 APJCP.pdf; Gelbke et al 2014.pdf; Zhang et al 2014.pdf; Wie te al 2014.pdf; dringen et al 2014.pdf; Sandvik et al 2014.pdf; Sapmaz et al 2015.pdf; Yu et al 2015.pdf

Dear Jason,

FYI

Look at the Gelbke one – that is relevant to your section. They are trying to dismiss the papillomas. The RAC would not give them the time of day so they wrote a paper to get it out there.

Ex. 5 Deliberative Process (DP)

Jane Caldwell

From: Caldwell, Jane
Sent: Friday, February 27, 2015 11:30 AM
To: Kraft, Andrew
Subject: FA literature

Dear Andrew,

I have been without a functional computer most of the week.

But today at least it is working.

My last lit update for Formaldehyde was in September 2014.

I am working on the MOA section again. I did a pubmed search to see what has shown up since my last search.

Here is a list of things that are out there that I could not download and need the HERO people to get if you have not already updated the lit again and have them. Not sure I can access HERO yet, have not tested that function on my computer since the screw ups all week.

I am also attaching things that are new that I already went and got. You can put them in your cache of papers and let the appropriate authors know about them.

Let me know if this is a problem.

Jane Caldwell

Literature to get PDF of – updated literature search from Fall 2014-March 2015

Peptides. 2014 Dec 22;64C:14-23. doi: 10.1016/j.peptides.2014.11.008. [Epub ahead of print]

Effect of carnosine supplementation on apoptosis and irisin, total oxidant and antioxidants levels in the serum, liver and lung tissues in rats exposed to formaldehyde inhalation.

Aydin S[1], Ogeturk M[2], Kuloglu T[3], Kavakli A[2], Aydin S[4].
Author information

Abstract
The main objective of the study has been to show whether carnosine has positive effects on liver and lung tissues of rats exposed to a range offormaldehyde concentrations, and to explore how irisin expression and antioxidant capacity are altered in these tissues by carnosine supplementation. Sprague-Dawley type male rats were divided into 8 groups with 6 animals in each: (I) Control; no chemical supplementation); (II) sham (100mg/kg/day carnosine); (III) low dose formaldehyde (LDFA) for 5 days/week; (IV) LDFA for 5 days/week and carnosine); (V) moderate doseformaldehyde (MDFA) for 5 days/week); (VI) MDFA for 5 days/week and carnosine; (VII) high dose formaldehyde (HDFA) for 5 days/week; (VIII) and HDFA for 5 days/week and carnosine. Sham and control groups were exposed to normal air. Irisin levels of the serum, liver and lung tissue supernatants were analyzed by ELISA, while the REL method was used to determine total oxidant/antioxidant capacity. Irisin production by the tissues was detected immunohistochemically. Increasing doses of FA decreased serum/tissue irisin and total antioxidant levels relative to the controls, as also to increases in TUNEL expressions, total oxidant level, oxidant and apoptosis index. Irisin expression was detected in hepatocyte and sinusoidal cells of the liver and parenchymal cells of the lung. In conclusion, while FA exposure reduces irisin and total oxidant in the serum, liver and lung tissues in a dose-dependent manner and increases the total antioxidant capacity, carnosine supplementation reduces the oxidative stress and restores the histopathological and biochemical signs.

G Ital Med Lav Ergon. 2014 Jul-Sep;36(3):153-9.

Exposure to formaldehyde in health care: an evaluation of the white blood count differential.

Sancini A, Rosati MV, De Sio S, Casale T, Caciari T, Samperi I, Sacco C, Fortunato BR, Pimpinella B, Andreozzi G, Tomei G, Tomei F.

Abstract
The aim of our study is to estimate if the occupational exposure to formaldehyde can cause alterations of leukocytes plasma values in health care workers employed in a big hospital compared to a control group. We studied employees in operating rooms and laboratories of Pathological Anatomy, Molecular Biology, Molecular Neurobiology, Parasitology and Experimental Oncology (exposed to formaldehyde) and employees of the Department of Internal Medicine (not exposed). The sample studied was composed of 86 workers exposed to formaldehyde and 86 workers not exposed. All subjects underwent a clinical-anamnaestic examination and for all subjects were measured the following values: total white blood cells, lymphocytes, monocytes and granulocytes (eosinophils, basophils, neutrophils). Statistical analysis of data was based on calculation of the mean, standard deviation and the distribution into classes according to the nature of each variable. Differences were considered significant when p was < 0.05. The mean and the distribution of values of the white blood cells, lymphocytes, monocytes and eosinophils were significantly higher in male subjects exposed to formaldehyde compared to not-exposed. Not significant differences were found in female subjects exposed compared to not exposed. The results underline the importance of a careful risk assessment of workers exposed to formaldehyde and the use of appropriate preventive measures. The health care trained and informed about the risks he is exposed to should observe good standards of behavior and, where it is not possible to use alternative materials, the indoor concentrations of formaldehyde should never exceed occupational limit values.

Neurobiol Aging. 2015 Jan;36(1):100-10. doi: 10.1016/j.neurobiolaging.2014.07.018. Epub 2014 Jul 18.

Age-related formaldehyde interferes with DNA methyltransferase function, causing memory loss in Alzheimer's disease.

Tong Z[1], Han C[2], Qiang M[2], Wang W[3], Lv J[3], Zhang S[3], Luo W[4], Li H[4], Luo H[4], Zhou J[5], Wu B[2], Su T[2], Yang X[6], Wang X[7], Liu Y[8], He R[9].
Author information

Abstract
Hippocampus-related topographic amnesia is the most common symptom of memory disorders in Alzheimer's disease (AD) patients. Recent studies have revealed that experience-mediated DNA methylation, which is regulated by enzymes with DNA methyltransferase (DNMT) activity, is required for the formation of recent memory as well as the maintenance of remote memory. Notably, overexpression of DNMT3a in the hippocampus can reverse spatial memory deficits in aged

mice. However, a decline in global DNA methylation was found in the autopsied hippocampi of patients with AD. Exactly, what endogenous factors that affect DNA methylation still remain to be elucidated. Here, we report a marked increase in endogenousformaldehyde levels is associated with a decline in global DNA methylation in the autopsied hippocampus from AD patients. In vitro and in vivo results show that formaldehyde in excess of normal physiological levels reduced global DNA methylation by interfering DNMTs. Interestingly, intrahippocampal injection of excess formaldehyde before spatial learning in healthy adult rats can mimic the learning difficulty of early stage of AD. Moreover, injection of excess formaldehyde after spatial learning can mimic the loss of remote spatial memory observed in late stage of AD. These findings suggest that aging-associated formaldehyde contributes to topographic amnesia in AD patients.

Biotech Histochem. 2015 Jan;90(1):69-78. doi: 10.3109/10520295.2014.954620. Epub 2014 Sep 16.

Protective effects of proanthocyanidin and vitamin E against toxic effects of formaldehyde in kidney tissue.

Bakar E[1], Ulucam E, Cerkezkayabekir A.
Author information

Abstract

We investigated possible effects of proanthocyanidin (PA) and vitamin E on damage to rat kidneys induced by formaldehyde (FA), using biochemical characteristics and light and electron microscopy. Male rats were divided into control, FA, PA and vitamin E treated groups. Kidney tissue was observed by light and electron microscopy. Bcl-2/Bax rate was measured using immunohistochemistry. Malondialdehyde (MDA) and total sialic acid (TSA) levels, superoxide dismutase (SOD), glutathione peroxidase (Gpx), catalase (CAT) and myeloperoxidase (MPO) activities were measured. We found that FA caused damage to the parietal epithelial layer of the glomerulus, mononuclear cell infiltration, membrane damage in renal tubules, pyknotic nuclei, hypertrophic cells in Henle's loop and tubules, and loss of renal tubule integrity. We also observed invagination of the nuclear membrane, irregularity of chromatin material and loss of mitochondrial cristae. We observed increased Bcl-2 and Bax immunostaining in the FA group, but the Bcl-2/Bax rate remained unchanged in FA, PA and vitamin E groups compared to controls. Tissue MDA and TSA levels, and CAT and Gpx activities were increased, and SOD and MPO activities were decreased by FA toxicity. We observed a protective effect of PA in tissue MDA and TSA levels and SOD activities, because there was no difference in the PA group compared to the control group. We investigated the antioxidant effects of PA and vitamin E and found protective effects of PA against apoptosis.

Mol Med Rep. 2014 Oct;10(4):1915-20. doi: 10.3892/mmr.2014.2473. Epub 2014 Aug 8.

Formaldehyde induces bone marrow toxicity in mice by inhibiting peroxiredoxin 2 expression.

Yu G[1], Chen Q[1], Liu X[1], Guo C[2], Du H[1], Sun Z[1].
Author information

Abstract

Peroxiredoxin 2 (Prx2), a member of the peroxiredoxin family, regulates numerous cellular processes through intracellular oxidative signal transduction pathways. Formaldehyde (FA)-induced toxic damage involves reactive oxygen species (ROS) that trigger subsequent toxic effects and inflammatory responses. The present study aimed to investigate the role of Prx2 in the development of bone marrow toxicity caused by FA and the mechanism underlying FA toxicity. According to the results of the preliminary investigations, the mice were divided into four groups (n=6 per group). One group was exposed to ambient air and the other three groups were exposed to different concentrations of FA (20, 40, 80 mg/m3) for 15 days in the respective inhalation chambers, for 2 h a day. At the end of the 15-day experimental period, all of the mice were sacrificed and bone marrow cells were obtained. Cell samples were used for the determination of pathology, glutathione peroxidase (GSH-Px) activity and myeloperoxidase (MPO) activity and protein expression; as well as for the determination of DNA damage and Prx2 expression. The results revealed an evident pathological change in the FA-treated groups, as compared with the controls. In the FA treatment group GSH-Px activity was decreased, while MPO activity and protein expression were increased. The rate of micronucleus and DNA damage in the FA-treated groups was also increased and was significantly different compared with the control, while the expression of Prx2 was decreased. The present study suggested that at certain concentrations, FA had a toxic effect on bone marrow cells and that changes in the Prx2 expression are involved in this process.

PMID:

25109304